PROSPECTIVE STUDY ON INDIGENOUS NEGATIVE PRESSURE WOUND THERAPY AFTER INNOVATIVE HYDRODEBRIMENT IN TREATMENT OF DIABETIC ULCER IN GRH, MADURAI

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CERTIFICATE

This is to certify that this dissertation entitled **" Prospective Study On Indigenous Negative Pressure Wound Therapy After Innovative Hydrodebriment In Treatment Of Diabetic Ulcer In GRH, Madurai"** at Government Rajaji Hospital, Madurai submitted by DR CHRISTEENA INDRANI J to the faculty of General Surgery, The Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MS degree (Branch I) General Surgery, is a bonafide research work carried out by him under my direct supervision and guidance.

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This is to certify that the dissertation entitled **" Prospective Study On Indigenous Negative Pressure Wound Therapy After Innovative Hydrodebriment In Treatment Of Diabetic Ulcer In GRH, Madurai"** is a bonafide research work done by **DR.CHRISTEENA INDRANI J,** Post Graduate Student, Department of General Surgery, MADURAI MEDICAL COLLEGE AND GOVERNMENT RAJAJI HOSPITAL, MADURAI, under the guidance and supervision of **DR.S.R.DHAMOTHARAN M.S, FIAGES** Professor Department of Surgery, MADURAI MEDICAL COLLEGE AND GOVERNMENT RAJAJI HOSPITAL, MADURAI.

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INTRODUCTION

Diabetes mellitus virtually affects every organ system in the body and it can be well said that “Knowing diabetes, is like knowing the entire human body”. The ancient physician, Aretaeus of Cappadocia (81- 138 AD) was the first to use the term diabetes. The word diabetes is perhaps derived from a Greek word signifying a “siphon”. In 1920, Fredrick Banting, Charles Best and John James McLeod first isolated insulin from the pancreas and named it “Isletin”.

The world is at present experiencing a pandemic of Diabetes Mellitus, particularly of type 2 or adult onset. The magnitude of the problem of diabetes is enormous. By 2030, there will be 366 million diabetics in the world, which is mainly due to longer life expectancy and change in dietary habits. India will in future have the largest number of diabetics. A majority of these patients will be in the age group of 35 to 45 years. Approximately, 15% of these patients will develop foot problems.

And 1% of these patients are likely to lose a limb due to some foot pathology or the other. However, a major blessing in Indian patients are mainly neuropathic- infective and not ischemic-infective. The latter are more difficult to treat when compared to the treatment of neuropathic ulcers.

It is a sad fact that, as of today, a regular foot examination and monitoring is not routinely practiced by our people. In fact, the routine policy is – "No complaints- No examination". However, by the time the patient complains of some symptoms, the pathology is advanced and foot salvage becomes extremely difficult. Early detection and attention to warning signals in the foot definitely can salvage the limb to a greater extent.

DIABETIC FOOT

The diabetic foot may be defined as a group of syndromes in which neuropathy, ischemia and infection lead to tissue breakdown resulting in increased morbidity and possible amputation.

WHO definition:

"The foot of a diabetic patient that has the potential risk of pathologic consequences including infection, ulceration and/or destruction of deep tissues associated with neurologic abnormalities, various degrees of peripheral vascular disease and or metabolic complication of diabetes in the lower limb".

DIABETIC FOOT-MORBID ANATOMY

A sound knowledge of anatomy is essential while treating diabetic foot complications, particularly infections. unless aggressively and adequately treated for infections," the patient pays through his foot' and may ultimately go home without it".

Peculiarities of the foot

"Foot is a highly complex design of nature,energy efficient, shock proof and has resilient biomechanism, adopted to weight bearing and locomotion on uneven surfaces". The foot is more peculiar so that it a vulnerable target in diabetes. it is the farther point from the heart and hence the commonest site for arterial insuffiency.it is the most dependent part of the body there by a favourite site for venous insufficiency.it is the maximum weight bearing part and hence there are multiple pressure points which are poor prospects for survival of free skin grafts.

The foot is vulnerable to injuries.it is also a common site for peripheral neuropathy,wherein the foot is vulnerable to physical and thermal injuries,small muscle atrophy and deformities as well as trophic changes.The foot is also prone to footwear related problems like blisters(new shoes),corn or callosities,ingrown toe nail,hallux valgus and

Hammer toe .due to sociocultural practices,there are problems related to bare foot walking.

Skin, nails and subcutaneous tissues of the foot

The dorsal skin is thinner (2mm thick),lax and can be pinched,while the plantar skin is thick (5mm) and cannot be pinched.The foot has a thick stratum corneum and a thin dermis.The skin is rich in sweat glands on the plantar skin.The dermis is bound to underlying fascia to improve grip and to prevent gliding or sliding.infections of sole tend to point to the dorsum,because of the thick plantar skin.

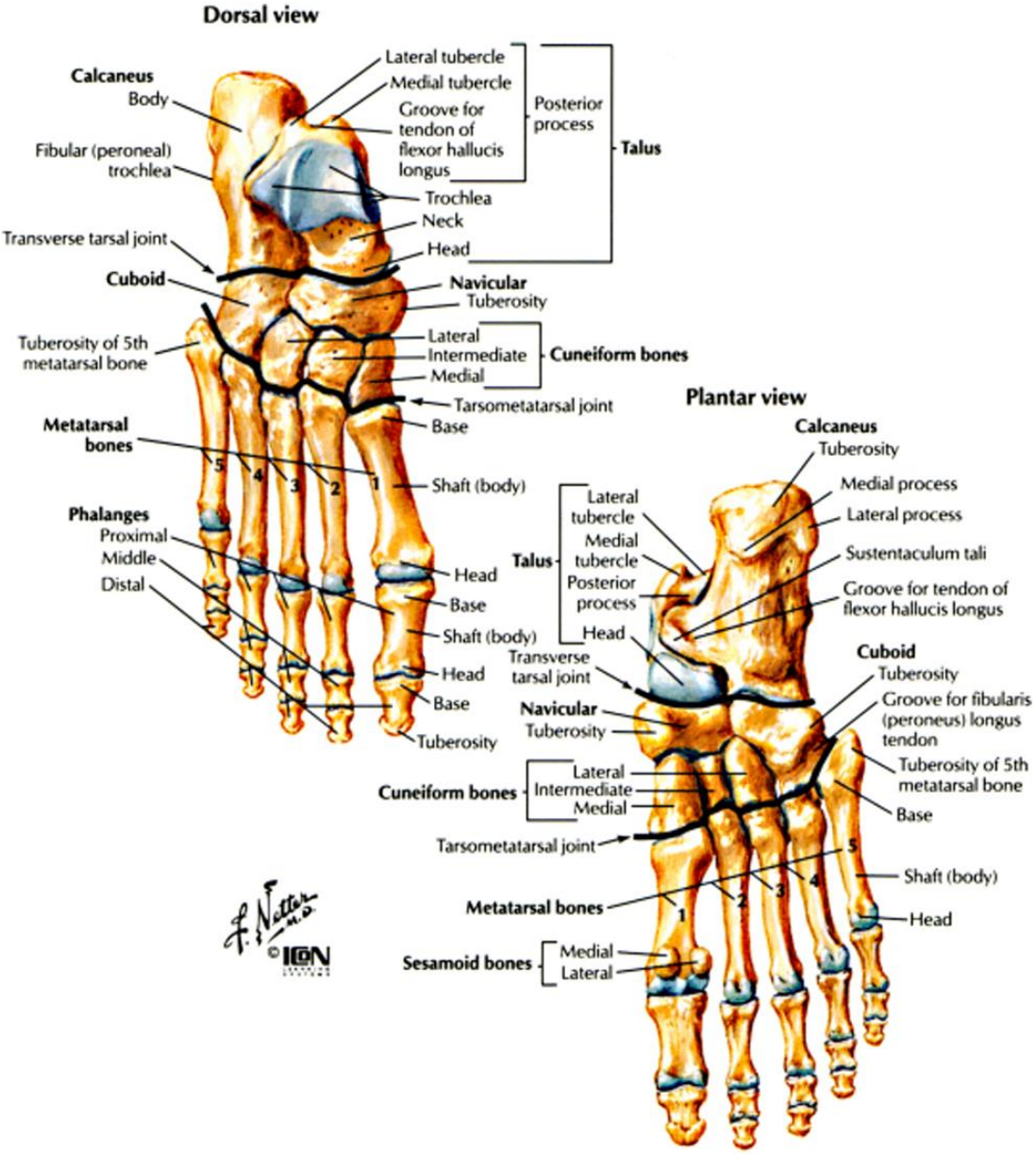
The epidermis gets transformed in to the nail matrix.it has three ill defined layers dorsal,intermediate and ventral layers.it is firmly attached to the epithelium of nail bed .the margins of the nail are overhung by skin folds predisposing to ingrown toe nails.The plantar subcutaneous tissue is more fibrous.The fluid fat is loculated by fibrous septa to provide shock absorption and to prevent gliding or sliding of plantar skin.

Skeleton and fascia of the foot

The skeleton of the foot is shaped to form arches and adjust to uneven surfaces.there are 7 tarsal bones,5 metatarsals and 14 phalanges.The superficial fascia of the sole is fibrous and dense.fibrous bands bind the skin to deep fascia or plantar aponeurosis.The fibrous bands divide the subcutaneous fat in to small compartments which serve as cushions and reinforce the spring effect of the arch during walking, running, jumping,etc.

The fascia is thick over weight bearing parts .it contains cutaneous nerves and vessels.The thickened central part of the deep fascia is the plantar aponeurosis.The plantar aponeurosis fixes the skin of the sole,protects deeper structures and helps in maintaining the longitudinal arches of the foot.it also gives origin to the muscles of the first layer of the sole.

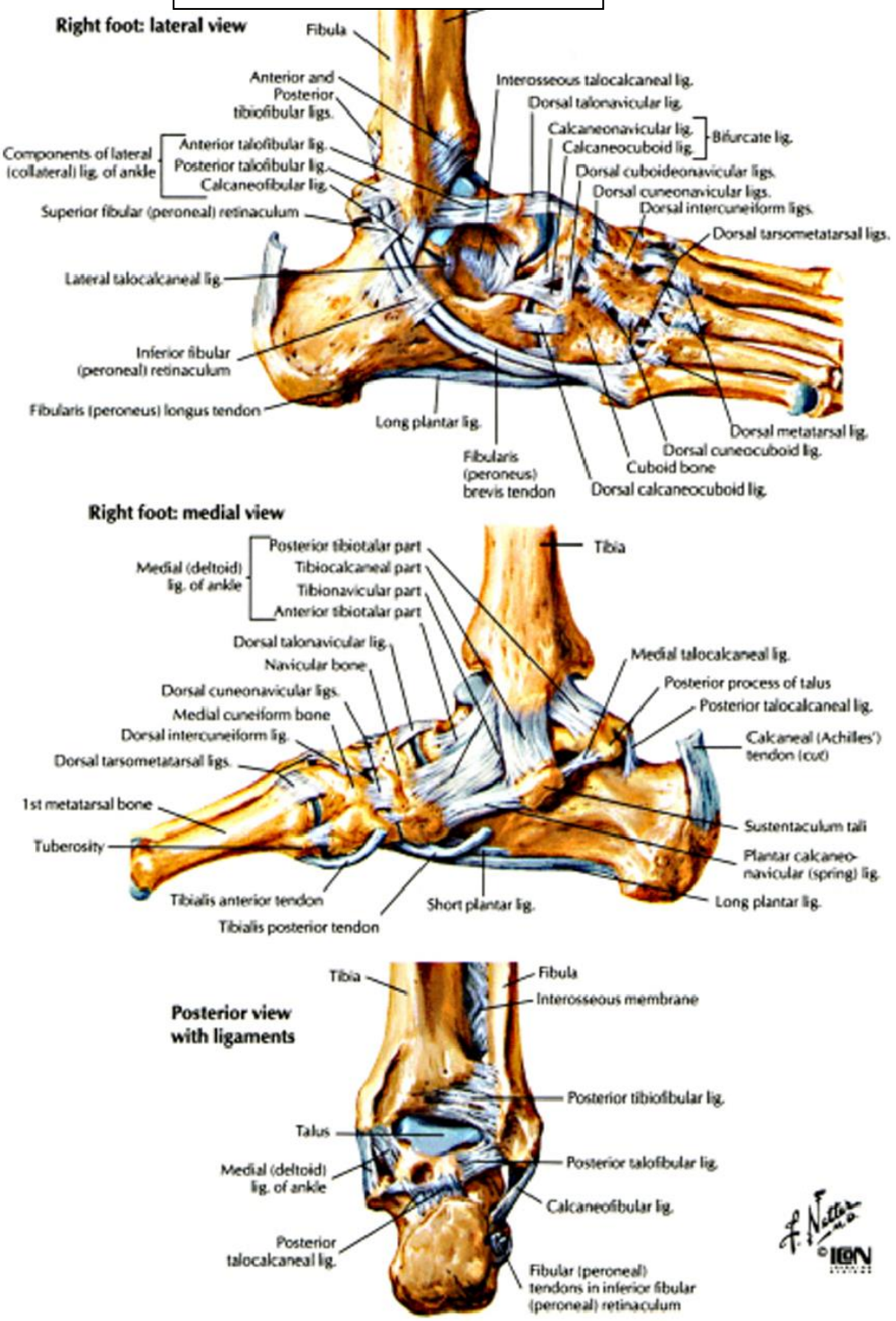
BONES OF THE FOOT



Ligaments of the foot

The ligaments maintain the arches and stability.they have a springing effect in locomotion and also help in shock absorption.The ligaments of the foot are long plantar ligament ,plantar calcaneocuboid (short plantar)ligament, plantar calcaneonavicular (spring)ligament, deltoidligament (medial), transverse metatarsal ligament, interosseous ligament.

LIGAMENTS OF THE FOOT



Muscles and tendons of the foot

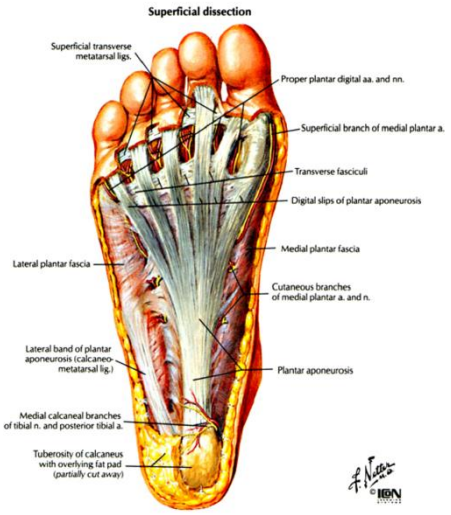
There are four layers which help in movement and grip and have a cushioning effect thereby protect nerves and vessels and they suspend arches. First layer includes abductor hallucis longus,flexor digitorum brevis,abductor digiti minimi .second layer is made of flexor accessorium (quadratus plantaris),tendons of flexor hallucis longus,flexor digitorum longus and the lumbricals.Third layer is constituted by the flexor hallucis brevis,transverse and oblique heads of the adductor hallucis,flexor digiti minimi brevis.the fourth layer is formed by the interrossei.

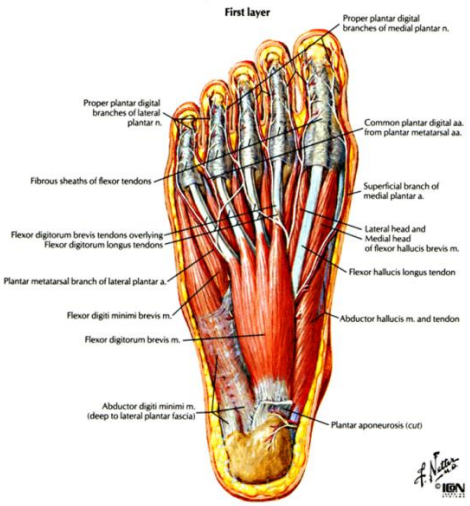
Musculo-fascial compartments of the foot

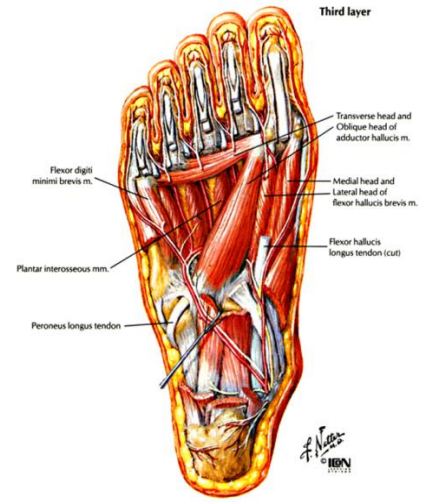
There are four compartments,formed by vertical septa from the plantar aponeurosis extending deep.they are the medial,central,lateral and interosseus compartments.

The medial compartment contains medial plantar nerve,artery,vein and the central(larger)compartment contains lateral plantar nerve,artery and vein.

LAYERS OF THE SOLE







Nerves of the foot

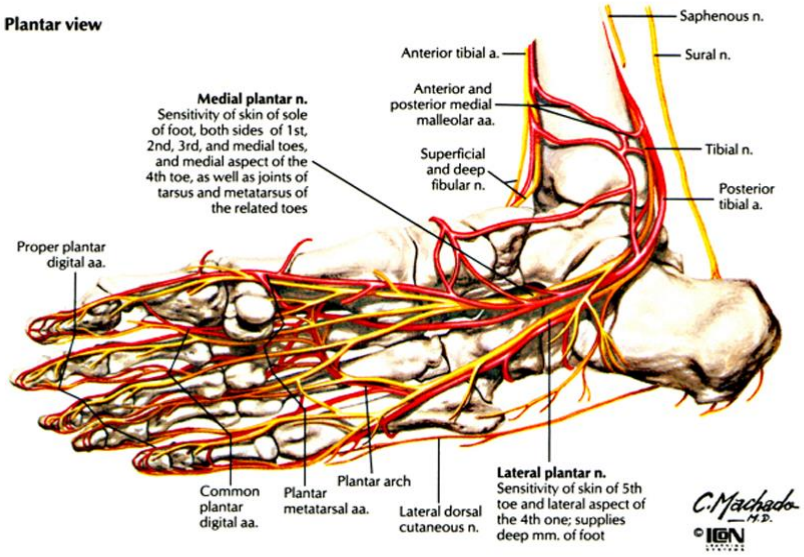
Saphenous nerve arises from the femoral nerve.it supplies medial aspect of the foot up to the first metatarsal.**Superficial peroneal (fibular)nerve** is the smaller terminal branch of the common peroneal nerve.it gives cutaneous branches to most of the dorsum of foot including digital branches to medial side of great toe,adjacent sides of second,third,fourth and fifth toes.**Deep peroneal (fibular)nerve** is the terminal branch of the common peroneal nerve.it supplies extensor digitorum brevis and gives cutaneous branch to the adjacent side of great and second toes.**Medial plantar nerve** is the largest terminal branch of the tibial nerve.it supplies abductor hallucis,flexor digitorum brevis,flexor hallucis brevis and first lumbrical muscle.cutaneous branches supply skin of the medial part of the sole and medial three and half toes.

Lateral plantar nerve is the smaller terminal branch of tibial nerve.The main trunk supplies flexor digitorum accessorius,abductor digiti minimi and skin of the sole.it divides in to superficial and deep branches.**sural nerve** arises from tibial and common fibular nerves and runs along the short saphenous vein.it supplies lateral side of the foot and fifth toe and all intrinsic muscles of the foot(S2 and S3).

Arterial tree

The dorsalis pedis artery is a continuation of anterior tibial artery and it runs between tibialis anterior and extensor hallucis longus tendons.it may be absent in about 5% of population.it gives arcuate artery,supplying the dorsum of foot and toes.The dorsalis pedis artery dips deep in the first inter-metatarsal space to form the plantar arch,by joining the medial and lateral plantar arteries.The posterior tibial artery ,runs behind the medial malleolus and divides in to medial and lateral plantar arteries,supplying the sole and toes.the plantar arch is formed by the medial and lateral plantar arteries with contribution from termination of the dorsalis paedis

artery. the digital arteries arise from the plantar arch (plantar aspect)and arcuate artery(dorsally).



Venous drainage

The dorsal venous arch lies in the dorsum of foot over the proximal parts of the metatarsal bones.it receives four dorsal metatarsal veins.These metatarsal veins are formed by the union of two dorsal digital veins.the long saphenous vein is formed by the union of the medial end of dorsal venous arch and the medial marginal vein.the medial marginal vein drains the medial side of the great toe.The short saphenous vein is formed by the union of lateral end of dorsal venous arch and lateral marginal vein.The lateral marginal vein drains the lateral side of the fifth toe.Both the

saphenous veins connect to deep veins through the perforating veins.

Lymphatic drainage

Superficial lymphatics drains along both the saphenous veins,short saphenous zone in to popliteal group and long saphenous zone in to inguinal group.Deep lymphatics drain along the arteries to both popliteal and inguinal groups.

Arches of foot

The arches help to adjust to uneven surfaces.The presence of arches makes the sole concave and this concavity protects the neurovascular structures.They are medial and lateral longitudinal arches and the anterior and posterior transverse arches.

Anatomical principles of surgical incisions

There are some anatomic principles one need to keep in mind while making incisions of the foot

Avoid neuro-vascular injury.

Avoid weight-bearing points.

Make liberal counter incisions

Deroofing should be liberal

Excise metatarsal head,while doing toe amputation for better realignment of toes.

DIABETIC FOOT - PATHOPHYSIOLOGY

Diabetes mellitus is associated with more than half of all non- traumatic lower limb amputations. The major pathophysiological factors are ischemia, neuropathy and wound infection. They operate concurrently and sequentially, enhancing the risk for amputation fifteen fold in diabetic subjects compared to non diabetics. Since the diabetic foot is the sequelae of interaction of a multitude of factors, intervention must be directed toward correction of all causative factors.

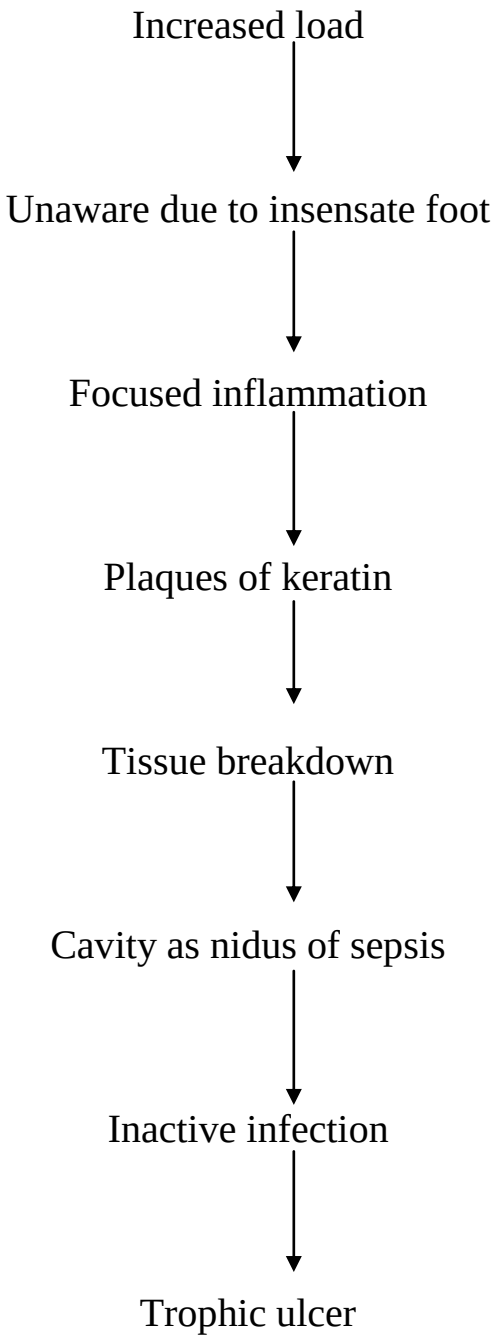
Diabetic Neuropathy

The most important factor leading to amputation for the person with diabetes is peripheral neuropathy and the resulting insensitive foot. Diabetic neuropathy affects sensory, autonomic and motor neurons of the peripheral nervous system, which is to say that every type of nerve fibre is affected. Diabetic peripheral neuropathy may be divided into two main types, acute sensory neuropathy and chronic sensorimotor neuropathy (most common).

Biochemical dysfunctions leading to neuropathy includes increased advanced glycosylation end products, defective polyol pathway, neurovascular alterations and impaired resistance to oxidative stress. The manifestations of sensory neuropathy are parasthesia, reduced pain perception, loss of joint sense, loss of vibration sense, glove and stocking anaesthesia, charcot joint. Motor neuropathy presents as weakness of muscles, paralysis of small muscles of foot producing deformed toes.

Autonomic neuropathy is characterized by the micro circulatory derangement of the tissues of the foot. There will be abnormal sweating and in some absence of sweating, dry foot with a lot of cracks in the sole, calcification of the mediumsized arteries and loss of thermoregulation.

Development of neuropathic ulcer



Diabetic macroangiopathy

Involvement of the major blood vessels is common in diabetes. The abdominal aorta and its branches are affected. The atherosclerosis is 20 times more common in the diabetics than the non diabetics. Calcification of the artery is also a common feature of diabetes. The media of the artery is calcified, which is called as Monckeberg sclerosis and this is due to neuropathy.

Calcification makes the vessel rigid and this gives a falsely elevated perfusion pressure. The crucial artery is the popliteal artery and its narrowing produces foot gangrene. Diabetes and smoking are the strongest risk factors for peripheral arterial disease. It is important to note that diabetes is most strongly associated with femoral- popliteal and tibial (below the knee) peripheral arterial disease.

Diabetic peripheral arterial disease has predilection for tibial and peroneal vessels but dorsalis pedis artery, the distal posterior tibial artery and the plantar arteries are usually spared. Diabetic patients with atherosclerotic peripheral vascular disease also show a diminished ability to establish collateral circulation.

Microvascular changes

Peculiar todiabetes is development of microvascular dysfuntion,which begins early in diabetic life.It is frequently seen in the capillaries and arterioles of kidneys,retina and peripheral nerves,but spares no organ.In diabetes RBC ‘s become flat and rigid and the blood is more viscous which are the major factor for microvascular impairment.Abnormalites in nitric oxide pathway, abnormal vasoconstrictor prostanoids, intracellular signalling, reduction in sodium –pottasium ATPase activity and advanced glycosylation end products are responsible for microvascular changes.

Vascular diseases:

The frequently associated risk factors for diabeti vascular disease include smoking, hyperlipidemia, insulin resistance with compensatory hyperinsulinemia, severity and duration of diabetes,age and generic factors. Smoking enhances the risk of peripheral vascular disease more than hundred times compared to non – diabetic non smokers.However ,cessation of smoking has been associated with a decrease in the progression of atherosclerosis.

Hypertension is twice as common in diabetics as compared to nondiabetics;roughly one third to one half of diabetics have hypertension.Systolic hypertension has been linked with disease of proximal blood vessels.

Diabetics and endothelial dysfunction

The mediator of endothelial cell dysfunction in diabetes is derangement of nitric oxide bioavailability.Nitric oxide inhibits vascular smooth muscle cell migration and proliferation and lilits platelet activation.Diabetes stimulates proatherogenic activity in vascular smooth muscle cells.

Diabetes,coagulation and rheology

Diabetes leads to hypercoagulable state .It is associated with the increase production of tissue factor by endothelial cells and vascular smooth muscle cells as well as increased plasma concentration of Factor VII.

Hyperglycemia is also associated with a decreased concentration of antithrombin and protein c ,impaired fibrinolytic function and excess production of plasminogen activator inhibitor.

Platelet aggregation is enhanced in diabetes.Platelet in diabetic patients also have increased expression of Glycoprotein IIb/IIIa receptors,which are important in thrombosis via their role in platelet adhesion and aggregation.

Infection

Infection is defined by invasion of the tissues with proliferation of microrganisms causing tissue damage with or without an associated inflammatory response by the host.Foot sepsis accounts for about 70% of all infections.Adherence of granulocytes and other WBC functions like phagocytosis are impaired in diabetes.T cell function is impaired and cell mediated immunity is depressed.Hyperkeratosis in foot is mistaken for a cornand removing it using rusted nail and safety pin is the formest reason leading to amputation.Absent sweating leads to cracks and fissures in foot which are portals of infection.Organism may be causative ,commensal ,contaminent orcoexisting polymicrobial..

Most common is polymicrobial infection.staphylococcus aureus and beta haemolytic streptococci are the most commonly involved pathogen in acute infection.In chronic wounds, Enterococci, Enterobacteriaceae,Obligate anerobes, Psuedomonas, Fungi are the pathogens involved.

Biomechanical aspects

Combination of neuropathy and trauma results in tissue breakdown.The atrophy of the intrinsic muscles of the foot,predominantly plantor flexors of the toes alters the flexor/ extension balance at the metatarsophalangeal joints and causes clawing of the toes and prominence of the metatarsal heads.

Alterations of the foot shape results in increased plantar pressure.A majority of wounds on insensitive foot are not caused by accidental injury or ischemia but from continuous pressure.

Often moderate stress as occurring during locomotion on the same part of the insensitive foot leads to callus formation and ulcer.The presence of callus may exacerbrate the problem both acting as a foreign body and by increasing the plantar pressure.

CHARCOT FOOT

Charcot foot or neuroarthropathy is defined as a relatively painless ,progressive,degenerative arthropathy of single or multiple joints caused by underlying neuropathy.Charcot neuropathy is characterized by simultaneous presence of bone and joint destruction,fragmentation and remodelling.Diabetes is the commenst cause of charcot foot and most patients have a dense neuropathy but good circulation.Walking on an insensitive foot leads to excessive and repetitive stress to bone causing micro fracture and finally bone and joint destruction

Diabetic neuropathy and presence of auto symphathectomy leads to peripheral vasodilation(warm foot).A Significant arteriovenous shunting takes place leading to abnormal bone cell activity (osteoclastic) and eventual resorption and weakening of bone.Ultimately the foot shape is deformed and runs into a bag of bones.Bone and joint damage in the metatarsal region is the commonest site of involvement and leads to the two classical deformities.

1.Rocker bottom deformities in which there is displacement and subluxation of the tarsus downward

2.Medial convexity ,which results from displacement of the talonavicular joint or from tarso-metatarsal disocation.

Both are often associated with a bony prominence which is very prone to ulceration.Healing is notoriously difficult.

If these deformities are not diagnosed early and accommodated in properly fitting footwear,ulceration at vulnerable pressure points often develops.It is not uncommon to mistake acute charcot foot for cellulitis and osteomyelitis.If the affected foot is elevated ,the erythema of charcot foot will recede whereas that for a cellulitis will persist.Acute charcot foot should not be mistaken for a cellulitis and operated.

Plain X ray of the foot will show demineralization,bone destruction and periosteal reaction.Marked osseous resorption of bone results in “pencil pointing “ and “sucked candy” deformities of the metatarsal heads and shafts.In the largest joints of the foot there will be destruction of bone and new bone formation.

It is a dictum that a “warm swollen foot in a diabetic with neuropathy without local and systemic signs of infection,charcot foot must be considered until proven otherwise”.

Osteomyelitis

Osteomyelitis should be suspected if the ulcer does not heal for more than 6 weeks of appropriate care and off loading ,and if there is swollen foot with ulcer,sausage toe,high WBC count or inflammatory markers.Radiographic evaluation is needed if osteomyelitis is suspected.Bone scan findings will be positive within 24 hrs while a plain X -ray will take 10 – 14 days to show any abnormality.There will be soft tissue swelling and periosteal elevation acute osteomyelitis and osteopenia, osteolysis and tapering of bones in chronic osteomyelitis.Any ulcer in which the bone is felt on probing with a sterile metal probe is likely to have osteomyelitis.

DIABETIC FOOT – CLINICAL ASSESSMENT

CLINICAL ASSESSMENT OF NEUROPATHY

1.Filament test: Semmes-Weinstein monofilament is used to detect the diminished sensation of foot.

Semmes –weinstein monofilaments

The monofilaments is a valuable and easy to use tool.the monofilament is a long nylon wire,the tip of which gives a force of 10 grams.It is pressed against the skin to the point of buckling for atleast one second.The points of testing are plantar aspects of 1^{st},3^{rd} and 5^{th} digits,the plantar aspect of 1^{st} ,3^{rd} and 5^{th} metatarsal heads,plantar mid foot medially and laterally and the plantar aspect of heel (10 sites totally).Neuropathy is said to exit when 4 out of these 10 sites show absence of sensation when the wire is pressed against the skin.

2.**Testing for vibration sense in toes and over the Malleoli**

Biothesiometer

This is also called as vibration perceotion threshold meter.This has a hand held probe whose tip vibrates at 100HZ.The probe is applied to a part of the foot, usually on the big toe.The probe can be made to vibrate at increasing intensity by turning a dial.The voltage supplied to the probe can be adjusted from 0 -50 V.The probe is placed against the skin and the voltage is increased until the patient perceives the vibration.Mean of three readings is used to determine the vibration perception threshold for each foot.Normal reading is less than or equal to 25V.

3.**Loss of joint position** is common in diabetic neuropathy.Joint sense of great toe is tested.Severe neuropathy produces small muscle wasting in the foot which leads to **collapse of arches** and deformity of toes.

These are precursors for ulcer formation.**Absence of sweating** makes the foot dry and prone for infection and cracks.Prominentlong saphenous vein is an index of **autonomic microcirculatory dysfunction.**This is a valuable clinical sign (J D Ward sign) of microcirculatory arterio venous shunting.

Clinical assessment of vascular disease

It starts with inspection of the foot for hue of toes,nicotine staining of fingers ,the thinning of skin due to loss of subcutaneous tissue and acral ulcers.Palpation of pulses (dorsalispedis,popliteal and femoral)remains the corner stone of screening for periphery vascular disease.Absence of distal pulse is a sure sign of significant arterial disease.However presence of palpable pulse does not absolutely exclude vascular disease.

Ankle brachial pressure index (ABI)

It is a simple method of assessing vascular insuffiency.It is obtained by dividing Ankle systolic pressure by Brachial systolic pressure.Normal values are 1+ 0.1.However ABI can be deceptive because calcification of vessels in diabetic can lead to falsely elevated ABI.All diabetics must have n annual assessment of ABI.

Indication for ABI monitoring

1.All those with type 1 diabetes older than 35 years or who have had diabetes for over 20 years at base line.

2.All those older than 40 years at base line with type 2 diabetes.

3.Any diabetic patient who has newly detected diminished pulses,femoral bruits or a foot ulcer.

4.Any diabetic with a leg pain of unknown etiology.

If ABI is more than 0.9 repeat every 2 -3 years.If ABI is 0.5 -0.89 repeat the measurement within 3 months and treat cardiovascular risk factors.If ABI is less than 0.5 ,refer for vascular work upand management.If an incompressible artery with an ankle pressure above 300mmhg or ankle pressure 75mmhg above arm pressure is found,measurement should be repeated in 3 months.If still present refer for vascular work up.

Infection

Infected ulcers are often asymptomatic in neuro ishemic foot of diabetics.The categorization of wound infection can be mild moderate and severe.Mild infections are superficial confined to the skin and subcutaneous tissue with minimal or no purulence or cellulitis.

Moderate infections are deep and may involve fascia,musles,tendons,joints and bones.They may present as cellulitis of less than 2 cm diameter,plantar abscess and with systemic symptoms.Severe infections are deep with cellulitis more than 2cm ,lymphangitis,gangrene and or necrotizing fasciitis, threatening limb loss and causing systemic toxicity.

INTEGRATED EXAMINATION OF DIABETIC FOOT

In practice the examination of the foot should be divided into four main parts:inspection,palpation,neurologicl examination and vascular assessment.

1.Inspection

The foot should be fully inspected including dorsum,sole,back of the heel and inter digital areas with full assessment regarding colour (asan indicator of ischemia),deformity ,swelling,callus,skin breakdown, infection ,necrosis.

2.Palpation

Pulse should be separated and skin temperature compared between both feet with the back of the examining hand.The measurement of the skin temperature is particularly helpful in the management of the charcot foot where a digital skin thermometer is useful.

3.Neurological examination

Peripheral neuropathy should be detected either by using the monofilament or biothesiometer or by performing a simple sensory examination.

4.Vascular status

All the peripheral pulses must be examined and compared with the normal limb.with regard to lowerlimb, femoral, popliteal, dorsalis pedis and posterior tibial arterial pulses must be examined.

DIABETIC FOOT – INVESTIGATIONS

LABORATORY TESTING

1.Blood sugar monitoring is the corner stone in management of any diabetic problem. A fasting and postprandial plasma glucose monitoring is essential in all diabetic foot patients.HBA1c monitoring is widely practised nowadays.

2.Bacterial culture

Superficial swabs are not useful. Necrotic tissue should be removed before taking a swab. Culture of swabs taken from the deeper part of the wounds will be effective in identifying the pathogens.

IMAGING STUDIES

Plain Radiographs are used to detect osteomyelitis, osteolysis, fractures, dislocations, etc. In a CT scan, the resolution of bone with osseous fragmentation and subluxation are well visualised. MRI aids in diagnosis of osteomyelitis, deep abscess, septic joint and tendon rupture. Three phase Technetium scans are used for early detection of osteomyelitis, fractures, charcot artheopathy . Indium III leucocyte scans and Tc 99 labelled white cell scan – differentiates osteomyelitis and neuropathic arthropathy.

Duplex ultrasound and Arteriography are used to detect arterial stenosis.

Gait analysis and Thermography

A walking cycle by definition is the time between the heel making contact with the ground and the same heel again coming in to contact with thw ground. In diabetes thickening of skin of sole due to abnormal weight bearing and infection under thick skin leads to altered gait. Neuropathy leading to claw toes and bent toes can lead to ulcers. Hence gait analysis in diabetics is mandatory for early detection of neuropathy.

Moderate repetitive stress with repetitive shearing force produces the typical neuropathic ulcer. Frictional forces occurring below the calcaneum is longitudinal while in forefoot it is both longitudinal and transverse. In a normal as well as in insensitive feet, walking briskly is accompanied by progressive hyperaemia over points of maximum stress. Thermography helps to outline the temperature contrast of progressive inflammation from such a process. In subjects with insensitive foot thermographic pattern shows hyperaemia at sites of old scar, there by inferring that these subjects have been stressing that particular area more than

optimally, due to absence of pain and as a result of motor neuropathy. Similarly, in – shoe foot prints help to detect the points of persistent and maximum stress on the feet which probably could be alleviated by proper footwear.

Harris mat

The feet can be evaluated for high pressure points by means of devices that can quantitate the pressure under the foot during walking or standing. A relatively inexpensive method to establish the presence of pressure points is the harris foot mat. This is an ink pad with graded depths of grid lines. The patient walks across the pad and the pressure points can be assessed by the intensity of the ink. This can also be fed in to a computer and a color coded analysis of pressure points can be obtained which is called as podio scan.

Plantar pressure measurement

Both the rear foot and fore foot pressures are elevated in diabetics. Restriction of movement of subtalar joint also leads to high plantar pressures. The areas of high pressure ultimately lead to tissue breakdown and ulcer formation. Computerised assessment of foot pressures is very important in assessing the changing pattern of

pressure transmission in the feet Plantar peak pressure more than 70N/cm2 is elevated.

Transcutaneous oximetry

It is measured at the dorsum of foot with patient in supine position. A transcutaneous oxygen tension of more than 55mmHg is normal, less than 40mmHg leads to failure of wound healing and less than 30mmHg predict limb loss.

CLASSIFICATION OF DIABETIC FOOT ULCERS

Wagner classification system

Grade	Lesion
0	No open lesions ; may have deformity or cellulitis
1	Superficial ulcer
2	Deep ulcer to tendon or joint capsule
3	Deep ulcer with abscess, osteomyelitis, or joint sepsis
4	Local gangrene – forefoot or heel
5	Gangrene of entire foot

University of texas classification system

Stage	Grade			
	0	1	2	3
A	Pre or post ulcerative lesions completely epithelized	Superficial wound not involving tendon,capsule,or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	Infected	Infected	Infected	Infected
C	Ischemic	Ischemic	Ischemic	Ischemic
D	Infected and ischemic	Infected and ischemic	Infected and ischemic	Infected and ischemic

MANAGEMENT OF DIABETIC FOOT

The foot ulcers in diabetics are not non healing ulcers but they are maltreated ulcers. Factors leading to wound healing deficiencies in diabetes are decreased or impaired growth factor production, angiogenic response, macrophage function, collagen accumulation, epidermal barrier function, quantity of granulation tissue, keratinocyte and fibroblast migration and proliferation, poor expression of matrix metalloproteinases and their inhibitors

a) WAGNER grade 0 foot:

This includes patients with apparently normal foot, varying degrees of neuropathy or joint deformities. They may not have any ulcer or infection but are potentially “at risk”. They need regular assessment atleast annually. Neuropathy must be looked for during each assessment. The best way to prevent neuropathy or delay it is to keep blood sugar under control.

Assessment of vascular status is also mandatory. Absent foot pulses even in the absence of claudication or rest pain indicates signicant vascular disease and such patients may be suitable candidates for vascular reconstruction or angioplasty. Remember that a diabetic may not manifest claudication symptoms if he had neuropathy. These “at risk” patients may have elevated pressure over some points on the sole. They need appropriate footwear (extradepth shoes with cushioned insoles). Charcot’s feet need custom shoes. Regular trimming of callus is needed. These patients also need advice regarding care of feet.

(b)**WAGNER grade 1 foot :**

These are patients who have presented with either cellulitis or a superficial ulcer. Ulcer occurs either with repetitive low pressure or sustained high pressure (>6kg/cm) at that point on the sole during walking.

Relief of pressure is the mainstay of ulcer treatment. An ulcer will not heal if the patient walks on it. A variety of ways are available to “off load” the ulcer. These include complete bed rest, use of total contact casts, walkers, braces etc. As in the case of

grade 0 feet, appropriate management of vascular disease is needed. Infection needs antibiotics and debridement as appropriate. Education, foot care and regular careful follow up are the principle factors in management of grade 1 foot.

(**c)WAGNER grade 2 and 3 foot :**

These are patients with deep ulcer with or without complications like abscesses and osteomyelitis. These patients need aggressive surgical debridement. Osteomyelitis must be appropriately managed by debridement/excision of infected bone.

Once the ulcer has healed, the patient needs long term care to advise appropriate foot wear and also education regarding foot care, in order to avoid recurrence.

(d)**WAGNER grade 4 and 5 foot :**

These are patients who have either localised or extensive gangrene. They need minor or major amputation respectively. Almost always there is vascular occlusive disease. These patients therefore need appropriate surgical amputation followed by vascular reconstructions. Aftercare involves special footwear for the ipsilateral and contralateral foot. (these patients tend to

over use the other foot and develop ulcers of the opposite foot). In case of major amputees, prosthetic devices need to be fitted in order to mobilize the patient. Mortality rate of diabetes after a major amputation is nearly 50% at one year.

Principles of medical management:

1. Pus from ulcers sent for pus culture and sensitivity.

2. Careful monitoring of blood glucose levels.

3. Appropriate antidiabetic measures – either insulin preparations or oral hypoglycemic drugs.

4. Broad spectrum antibiotics to be started at the onset and change over to other antibiotics depending on culture and sensitivity report.

Principles of surgical management:

1. Early recognition and prompt intervention.

2. Control of blood glucose.

3. Complete rest of injured area.

4. Careful but complete debridement and drainage of all involved areas.

5. Appropriate antibiotic coverage.

6. Wound care and dressings.

7. Appropriate vascular reconstruction.

8. Careful follow up including podiatric appliances and modified footwear.

9. More experienced consultation as necessary

VARIOUS TREATMENT MODALITIES

1.**Protective dressing**

Modern moist dressings include foams, calcium alginates, hydrogels, hydrocolloids, and adhesive membranes

2.**Topical antiseptics**

Superoxide solutions are useful topical antiseptics which are active against many organisms

3.**Drainage of pus**

Vaccum assisted drainage (continuous negative pressure of 125 mmHg) to the wound will promote healing of the ulcer.

4.**Debridement**

Aggressive ongoing surgical debridement converts a chronic non healing ulcer in to an acute healing wound.

Adequate debridement of necrotic tissue (eschar, slough) is needed before adequate assessment and staging can be accomplished. There are several methods of wound debridement, including sharp surgical, mechanical, enzymatic and autolytic. It is continuum from flushing away debris with low pressure irrigation to wide excision.

Sharp surgical debridement

The most selective and efficacious method of debridement is sharp surgical debridement. Debridement of the hyperkeratotic rim and ulcer base to bleeding

is the optimal method of debridement for the patient with an ulcer.

Autolytic debridement

Autolytic debridement with moist interactive dressings (hydrogel,alginates,transparent films,hydrocolloids) is selective and liquefies slough and eschar as well as promotes granulation tissue formation.

Mechanical debridement

Mechanical debridement may be accomplished with wet to dry gauze dressings, irrigation, pulsatile lavage or whirl pool

Enzymatic debridement

Historical enzymes (collagenase , papain, urea) have been used as debriding agents for eschar and slough. They have a selective action, but are slow, costly and labour intensive.

5. Antibiotic

The use of double or triple antibiotics seems to be justified. The antibiotics used must have broader spectrum covering gram positive, gram negative and anaerobic organisms.

MANAGEMENT OF INDOLENT NON HEALING ULCERS

A.Collagen sheets and powders

B.Silver dressing

C.Growth factors

Growth factors are derived from platelets, bioengineered tissues or by recombinant techniques.

NEGATIVE PRESSURE WOUND THERAPY & HYDRODEBRIDEMENT

NPWT is a novel technique for managing an open wound by submitting the wound to either intermittent or continuous subatmospheric pressure. Negative pressure is obtained by transferring away the gas molecules that are present in the wound away by using a suction pump.

There are two types of NPWT in practice now:

1) Foam based technique

2) Chariker Jeter Technique

FOAM BASED TECHNIQUE:

Here, a foam cut in the shape of the ulcer is placed over te wound and sealed in place using an adhesive film drape and a TRAC (Therapeutic Regulated Accurate Care) system. Plastic tubing is used to connect the dressing to the console that applies the suction force.

CHARIKER JETER TECHNIQUE:

It is a more recently developed system that uses flexible drains and moist gauze. The moist gauze is placed in the wound after insertion of a silicone drain and sealed using an adhesive film drape creating an airtight seal. The silicone drain is attached to the suction console.

MECHANISM OF ACTION:

- Increased blood flow to wound bed.
- Increased neovascularization with profuse granulation tissue.
- Increased activity of fibroblasts.
- Increased epithelial cell migration.
- Decreased bacterial toxins.
- Decreased harmful wound fluid and toxic products.
- Decreased microvascular occlusion and inelasticity.
- Decreased periwound induration.
- Reduction in number of dressing changes → decreased damage to delicate new tissue.
- Provision of healing by primary intention by mechanical approximation.

- Promotes epitheliasization and synthesis of growth factors.
- Persistent stimulation of cytoskeleton → increased mitosis.
- Increased viscoelastic flow due to stretching of tissues.
- Decreased shear forces due to uniform wound bed immobilization.
- Decreased seroma of grafts and flaps.
- Splinting effect (sternal, abdominal wound)

Contrindications to NPWT:

- Exposed vital organs
- Contaminated wounds
- Untreated osteomyelitis.
- Untreated coagulopathy.
- Necrotic tissue with eschar.
- Malignancy in the wound.
- Allergy to any component involved in the procedure.

NPWT should be used with extreme caution if there is active bleeding, if the patient is on anti-coagulants and if the dressing is in close proximity to blood vessels.

If there is any exposed vessel or vital organ, petroleum impregnated gauze should be interposed between vital structure and sponge.

HYDRODEBRIDEMENT:

Hydrodebridement includes the use of a high pressure waterjet oriented parallel to the surface of the wound capable of tangentially excising soft tissues at variable strengths. It works on the Venturi effect, a special case of Bernoulli's principle, which states that a fluid flowing through a tube that contains a constriction must increase the velocity in order to decrease the pressure and still maintain the conservation of energy. This procedure eases the method of debridement selectively removing slough and debris and thus reduces the bacterial counts in contaminated wounds.

MATERIALS AND METHODS

AIM & OBJECTIVE

To study the effect of indigenous negative pressure wound therapy after innovative hydrodebridement in treatment of diabetic ulcer in GRH, Madurai.

INCLUSION CRITERIA

- Patients more than 25 years of age groups in both sexes presenting with diabetic ulcer.
- Patients consented for inclusion in the study according to designated proforma

EXCLUSION CRITERIA

- Patients less than 25 years of age.
- Osteomyelitis.
- Unexplored fistulas.
- Overexposed blood vessels.
- Unstable general condition.
- Patient not consented for inclusion in the study.

STUDY AREA

Govt Rajaji Hospital, Madurai.

STUDY PERIOD

July 2016 to September 2017

SOURCE OF DATA

All patients diagnosed to have diabetic ulcer, who also come under the inclusion criteria.

METHOD OF COLLECTION OF DATA

Details of cases, Full history, Clinical Examination, Dimensions of the ulcer, Rate of granulation tissue formation, Duration of hospital stay until the wound is fit for grafting.

METHODOLOGY:

Patients selected according to the criteria included in this study were subjected to hyrodebridement using a conventional suction- irrigation unit, wherein the irrigation pressure could be adjusted manually. This was done on a thrice weekly basis or during intervals of NPWT or as and when required depending on the amount of slough.

After HD, a drain tube was placed and fixed inside the wound. A foam cut according to the shape of the ulcer was placed and an airtight seal was created using adhesive tapes. The drain was then connected to the suction unit and intermittent suction was applied at 3 hour intervals.

Dressing was changed once in two days or according to the amount of exudate.

Reduction in ulcer surface area, rate of granulation tissue formation, uptake of SSG and duration of hospital stay were assessed and results were obtained.

OBSERVATIONS AND RESULTS

The 100 patients admitted for the study were divided into two equal and comparable groups. Patients subjected to indigenous negative pressure wound therapy after innovative hydrodissection were classified under study and those who underwent conventional moist wound dressing were classified as control.

Table: Sex wise distribution of patients.

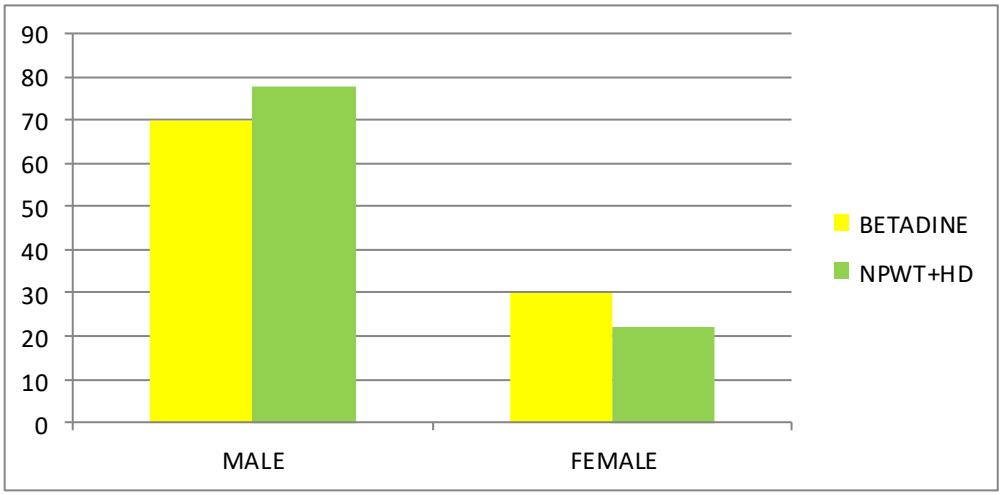


TABLE: Age wise distribution of patients

AGE GROUP (yrs)	31-40	41-50	51-60	61-70	71-80
BETADINE	1	14	20	12	3
NPWT + HD	6	10	21	9	4
TOTAL	7	24	41	21	7

Mean age of Betadine group is 56.12 + 8.76.

Mean age of NPWT + HD group is 54.54 + 11.003.

P value is 0.429. Not significant.

TABLE : Age wise distribution of patients

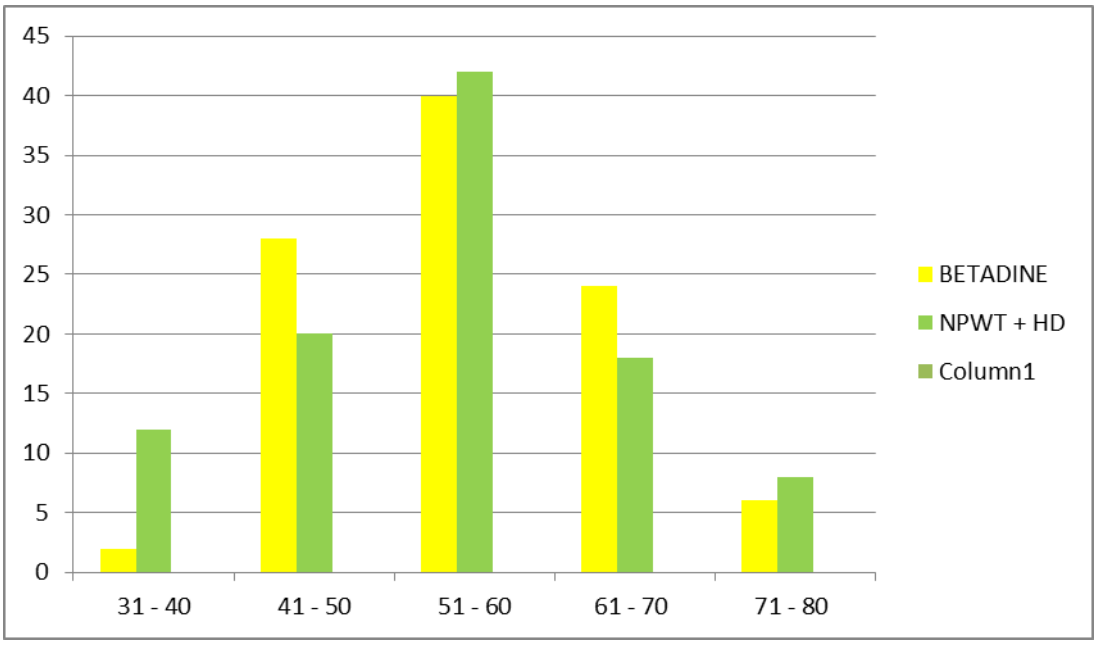
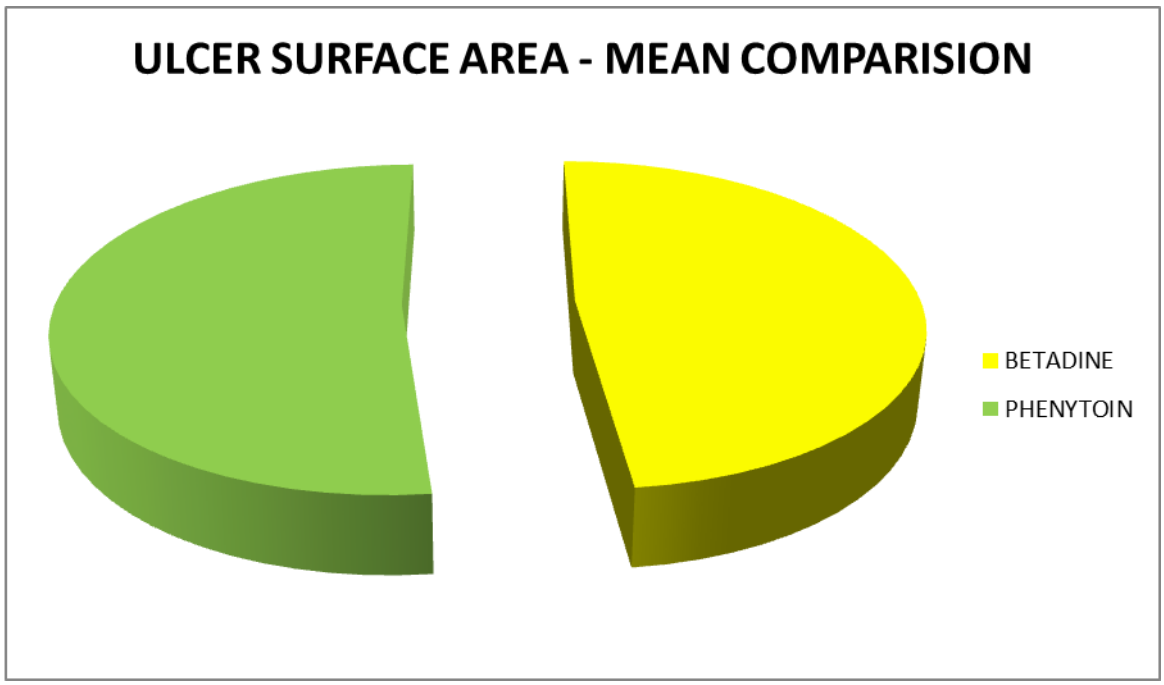


TABLE: ULCER SURFACE AREA

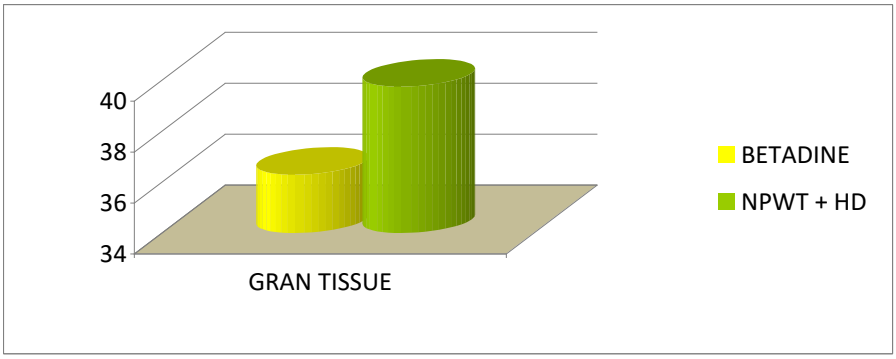
GROUP	N	MEAN	STD. DEVIATION	MEDIAN	t VALUE	p VALUE
BETADINE	50	37.67	7.28	38.66	2.509	0.014
NPWT + HD	50	40.44	2.88	40.41		sig



The mean ulcer surface area in control group is 37.67 cm^2 and in the study group is 40.4 cm^2 . The ulcer surface area is measured twice using butter paper.

TABLE: Rate of granulation tissue formation as percentage of ulcer surface area

GROUP		NO.	MEAN	STD. DEVIATION	MEDIAN	t VALUE	p VALUE
GRAN TISSUE	BETADINE	50	36.29	5.82	37.36	3.783	<0.001
	NPWT + HD	50	39.76	2.84	39.65		HS

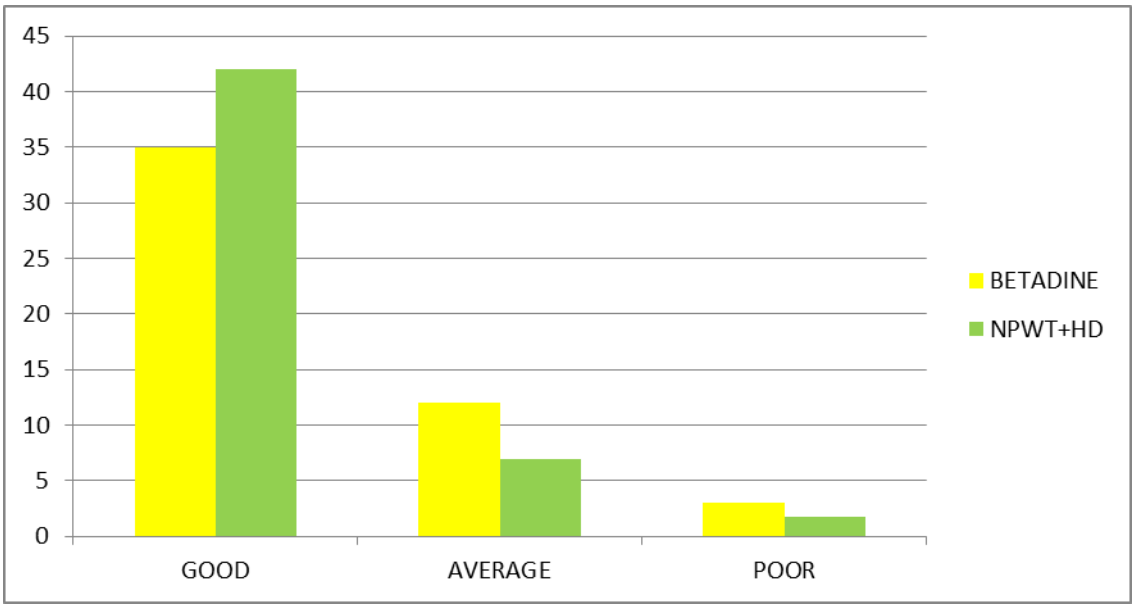


The mean rate of granulation tissue formation in

Betadine group is 36.29cm2 ± 5.82 (SD) of total ulcer surface area and in NPWT + HD is 39.76 ± 2.84 (SD) of total ulcer surface area.

TABLE: GRAFT UPTAKE AS PERCENTAGE OF ULCER SURFACE AREA

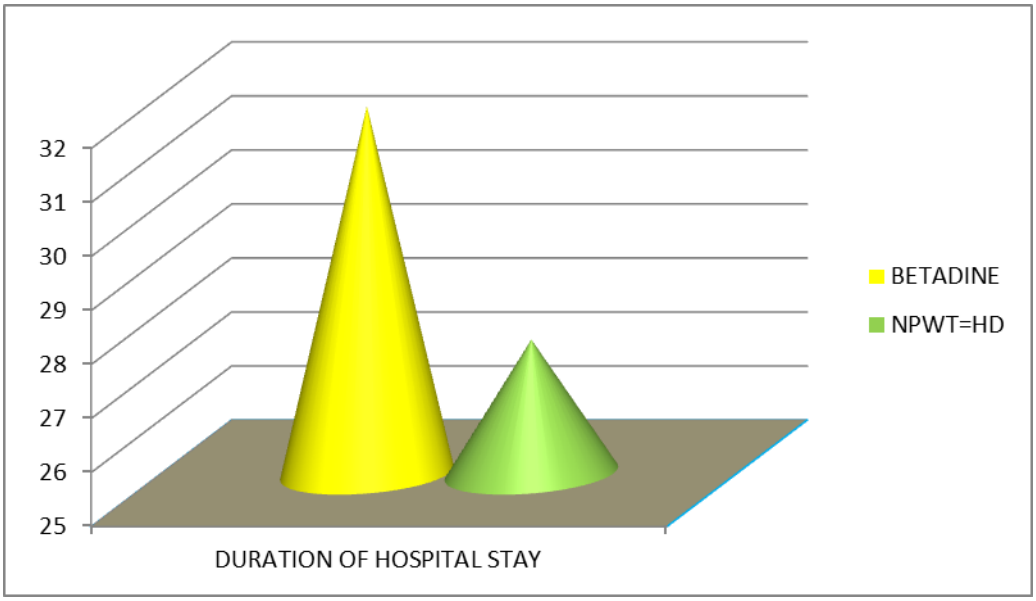
GROUP		N	GOOD	AVERAGE	POOR	MEAN % OF GRAFT UPTAKE
STSG	**BETADINE**	50	35	12	3	84%
	NPWT + HD	50	42	7	1	70%



Assessment of graft uptake was done at the end of POD 5 as percentage of ulcer surface area. The mean graft uptake in the study group is 37.35% ± 5.64 (SD) and in the control group is 40.37% ± 2.83 (SD).

TABLE: DURATION OF HOSPITAL STAY

GROUP		N	MEAN	STD DEVIATION	MEDIAN	t VALUE	p VALUE
NO OF DAYS	BETADINE	50	31.80	4.63	30.00	5.7.6	<0.001
	NPWT + HD	50	27.48	2.68	28.00		HS



The quality of life of the patient in both the groups was assessed by the assessment of total hospital stay as number of days of admission in the hospital. The mean hospital stay in the control group was 31.8 +4.63 (SD) days and that in the study group was 27.48 + 2.68 (SD) days. P value is <0.001 which is highly significant.

The main postoperative parameters noted in both the groups:

- Wound size
- Contracture
- Pain
- Infection

All these parameters were less in the study group when compared with the control group.

ANALYSIS OF DATA:

Both the groups had comparable age and sex distribution as depicted in the graphs above.

The mean rate of granulation tissue formation in study group is 95.93 cm^2 of total ulcer surface area and in control group is 98.09cm^2.the results were analyzed by unpaired student t test which showed highly significant difference in the rate of granulation tissue formation ($p<0.0002$). The mean graft uptake in the study group is 99.03cm^2 and in the control group is 97.61cm^2. The results were analyzed by unpaired student t-test which showed highly significant difference in graft uptake (p of 0.001). The total number of days of hospital stay was also compared. The mean number of days of hospital stay in the control group was 31.3 and that in the study group was 27.8 days. The results were analyzed by unpaired student t-test which showed highly significant difference in the number of days of hospital stay ($p< 0.0002$).

FIG: HYDRODISSECTOR UNIT

FIG: SUCTION UNIT

FIG: DIABETIC ULCER ON PRESENTATION

FIG: HYDRODEBRIDEMENT

FIG: SUCTION DRAIN IN SITU WITH FOAM PLACED ON ULCER BED

FIG: ADHESIVE DRESSING

FIG: DIABETIC ULCER ON PRESENTATION

FIG:SUCTION DRAIN INSITU

FIG. ADHESIVE DRESSING

FIG – NPWT IN ACTION

FIG: WOUND AFTER NPWT + HD

FIG: WOUND AFTER SSG

CONCLUSION

- NPWT + HD significantly reduces the size of ulcer.
- NPWT + HD improves the rate of granulation tissue formation.
- NPWT + HD improves SSG uptake also.
- NPWT + HD reduces the duration of stay at the hospital.
- Patients undergoing hydrodebridement undergo lesser amount of pain when compare to patients undergoing conventional wound debridement.
- Hydrodebridement minimizes the blood loss also.

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PROFOMA

Name:

Age:

Sex:

IP No:

Date of admission:

Date of discharge:

Duration:

DM: Y/N

Alcoholic: Y/N

Co- morbid medical conditions:

History of present ulcer:

Examination of ulcer:

Site:

Size:

Shape:

Margin:

Floor:

Base:

Vascular status:

Skin:

Pulse:

Investigations:

Hb: TC: DC: Blood Group:

FBS/PPBS:

Blood urea/ Sr creatinine:

ECG:

Doppler profile:

Xray:

Viral markers:

Treatment:

Antibiotics (if any):

Insulin dosage:

Any OHA:

Treatment before NPWT + HD:

NPWT + HD:

No of application:

S No.	DATE OF APPLICATION	DATE OF REMOVAL

After NPWT + HD:

Granulation tissue: Y/N

Exudate:

Plan after NPWT + HD:

S No	NAME	AGE	SEX	IP No	DOA	DOD	NO OF DAYS OF HOSPITAL STAY	TYPE OF DRESSING	ULCER AREA	GRAN AREA	SSG
1	VELUCHAMY	38	M	40459	7/14/2016	8/17/2016	33	B	40.32	37.6	45
2	SELVAM	52	M	32118	7/20/2016	8/27/2016	37	B	42.73	39.1	30
3	SELVARAJ	65	M	4076	7/22/2016	8/26/2016	35	B	37.72	37.16	39
4	MAHESHWARI	65	F	55994	7/8/2016	8/18/2016	41	B	43.69	40.7	70
5	HARIHARAN	62	M	34210	7/11/2016	8/23/2016	43	B	44.5	43.83	78
6	ARUMUGAM	58	M	3813	8/22/2016	9/21/2016	30	B	37.14	35.9	75
7	MUTHUPECHI	52	F	37155	8/5/2016	9/6/2016	31	B	36.79	34.7	67
8	PUSHPA	55	F	38547	8/16/2016	9/15/2016	29	B	40.1	39.15	56
9	MUNISWARI	70	F	32327	8/29/2016	9/31/2016	32	B	43.52	41.84	57
10	SURESH	50	M	32330	9/7/2016	10/10/2016	34	B	38.16	36.43	59
11	FATHIMA	55	F	32334	9/12/2016	10/19/2016	38	B	41.65	41.1	69
12	VANITHA	40	F	32337	9/20/2016	10/18/2016	29	B	38.43	37.64	81
13	JOSEPH	48	M	32343	9/5/2016	10/14/2016	40	B	44.1	42.8	78
14	KASIRAM	42	M	32346	10/13/2016	11/14/2016	32	B	37.18	35.9	75
15	RAM	62	M	32350	10/23/2016	11/27/2016	35	B	39.6	37.12	56
16	DHARMAR	60	M	32353	10/6/2016	11/8/2016	30	B	38.7	37.15	76
17	ISAMMA	65	F	32358	10/16/2016	11/20/2016	32	B	42.17	41.5	72
18	SAROJA	55	F	32361	11/20/2016	12/22/2016	30	B	43.84	41.9	71
19	MUTHUPECHI	45	F	32369	11/6/2016	12/13/2016	38	B	51.18	45.44	54
20	SEKHAR	72	M	32374	11/10/2016	12/11/2016	32	B	30.96	22.32	51
21	NARAYANAN	76	M	32396	11/17/2016	12/11/2016	25	B	44.36	44.24	50
22	IMRAN	72	M	32404	12/2/2016	12/30/2016	28	B	33.72	30.36	82
23	USMAN MOHD	58	M	32419	12/10/2016	12/22/2016	42	B	39.3	34	89
24	JOHN	54	M	32431	12/15/2016	1/15/2017	30	B	25.3	23.6	90
25	KUMAR	46	M	32452	12/7/2016	1/4/2017	28	B	29.45	26.15	81
26	VINCENT	54	M	1024630	1/11/2017	2/16/2017	26	B	32.6	30.15	80
27	MUNIYAMMA	65	M	1024871	1/21/2017	2/15/2017	25	B	26.32	24.19	95
28	KUMARASAMY	42	M	1025142	1/7/2017	2/5/2017	28	B	29.1	27.6	97
29	MALAISAMY	58	M	1025491	1/8/2017	2/4/2017	26	B	42.4	39.16	82
30	SUBRAMANI	60	M	1025392	1/18/2017	2/30/2017	42	B	45.5	40.76	79
31	KALYANI	58	F	10256131	2/2/2017	3/1/2017	30	B	36.18	35.43	81
32	AKBAR PASHA	49	M	1025886	2/21/2017	3/18/2017	28	B	39.49	37.SS	89
33	HUSSAIN	54	M	1026093	2/5/2017	3/2/2017	29	B	32.39	30.19	20
34	MARY	45	F	1026172	2/15/2017	3/11/2017	28	B	28.72	25.73	27
35	KHATHIJAMMA	58	F	1026490	2/3/2017	3/8/2017	35	B	30.36	28.54	67

36	SUNITHA	62	F	1026672	3/19/2017	4/19/2017	30	B	37.4	39.52	34
37	SOMASUNDARAM	68	M	1027106	3/12/2017	4/11/2017	30	B	38.6	34.35	35
38	KRISHNAN	54	M	1027491	3/20/2017	4/21/2017	32	B	43.76	43.26	39
39	VEERAPPAN	55	M	1028164	3/4/2017	4/4/2017	30	B	39.7	37.63	40
40	MANIKANDAN	45	M	1028570	3/17/2017	4/13/2017	26	B	42.6	40.17	42
41	JANAKI	60	F	1028911	4/5/2017	5/3/2017	28	B	32.26	29.69	43
42	MATHEW	64	M	1029073	4/22/2017	5/23/2017	32	B	36.42	33.27	32
43	GANESAN	48	M	1029145	4/10/2017	5/7/2017	28	B	35.42	32.9	21
44	PERIYASAMY	47	M	1029221	4/23/2017	5/28/2017	28	B	36.72	35.2	29
45	BABU	48	M	1029238	4/7/2017	5/10/2017	35	B	38.62	37	39
46	JEYAMANI	52	M	1029290	5/14/2017	6/14/2017	32	B	40.92	39.2	42
47	MOHD SHEIK	50	M	1029312	5/11/2017	6/18/2017	38	B	44.6	42.72	39
48	RAJENDIRAN	58	M	1029329	5/25/2017	6/24/2017	30	B	41.9	40.25	48
49	CHELLAMMAL	62	F	1029345	5/2/2017	5/30/2017	28	B	42.7	41.2	42
50	MANIMARAN	68	M	1029367	5/12/2017	6/14/2017	32	B	4.16	41.28	43
51	PALRAJ	52	M	32301	7/5/2016	8/5/2016	30	H & N	39.42	37.6	100
52	HARIHARAN	38	M	32304	7/15/2016	8/8/2016	23	H & N	41.15	39.69	95
53	VIKRAM	50	M	32308	7/22/2016	8/19/2016	27	H & N	43.S	41.9	94
54	KAMALA	60	F	32317	7/9/2016	8/4/2016	26	H & N	35.28	36.69	89
55	ANDIAPPAN	58	M	32318	7/12/2016	8/4/2016	23	H & N	37.49	36.8	88
56	MANGALAPANDIAN	65	M	32319	8/22/2016	9/13/2016	21	H & N	39.78	39.S	88
57	KALASIAMMAL	62	F	32324	8/7/2016	9/9/2016	32	H & N	44.63	44.75	90
58	JEGANATHAN	55	M	32325	8/15/2016	9/15/2016	30	H & N	42.S	41.85	86
59	RAMAYEE	52	F	32326	8/23/2016	9/21/2016	28	H & N	43.72	47.1	83
60	VIGNESHWARI	52	F	32334	9/12/2016	10/4/2016	23	H & N	36.8	36.15	79
61	CHELLAPANDIAN	62	M	32335	9/15/2016	10/9/2016	25	H & N	38.14	37.84	89
62	SHEIK MOHD	64	M	32338	9/24/2016	10/23/2016	30	H & N	39.4	38.6	97
63	PRAKASHRAJ	38	M	32341	9/3/2016	9/30/2016	28	H & N	40.14	39.S	96
64	NAGESH KUMAR	57	M	32342	10/5/2016	11/4/2016	30	H & N	42.73	41.96	93
65	SHAHUL KHAN	52	M	32348	10/17/2016	11/14/2016	28	H & N	43.16	42.8	92
66	XAVIER	42	M	32337	10/7/2016	11/3/2016	28	H & N	42.52	41.19	91
67	JOSEPH	60	M	32353	10/13/2016	11/7/2016	26	H & N	40.69	39.35	87
68	CHOKKALINGAM	52	M	32359	11/16/2016	12/9/2016	25	H & N	36.7	35.49	89
69	ABDUL SALEEM	36	M	32359	11/5/2016	12/28/2016	23	H & N	38.46	37.25	88
70	MUNIYASAMY	69	M	32361	11/15/2016	12/20/2016	26	H & N	37.41	36.S	85
71	SENTHIL	40	M	32376	11/23/2016	12/19/2016	27	H & N	42.79	41.8	84
72	VAIRAVAN	32	M	32379	12/4/2016	12/29/2016	25	H & N	38.16	38	88
73	IRULAYEE	40	F	32397	12/17/2016	1/14/2017	27	H & N	40.53	39.61	82
74	YASHODAKUMARI	42	F	32406	12/22/2016	1/20/2017	28	H & N	36.7	36.1	83
75	MUNIYASAMY	62	M	32420	12/2/2016	1/28/2017	26	H & N	43.25	42.62	81
76	SETHURAMAN	75	M	1045715	1/7/2017	2/6/2017	30	H & N	40.29	39.84	80
77	MUTHUPANDI	60	M	1024691	1/12/2017	2/7/2017	26	H & N	37.2	36.9	89

78	VASUKI	72	F	1024790	1/15/2017	2/16/2017	32	H & N	45.61	43.7	88
79	MOHD ALI	79	M	1024915	1/26/2017	2/21/2017	25	H & N	35.43	35.16	85
80	MARIMUTHU	42	M	1025312	1/27/2017	2/25/2017	28	H & N	44.64	43.37	87
81	PALRAJ	70	M	1025512	2/9/2017	3/10/2017	32	H & N	46.7	44.16	82
82	RAMARAJAN	68	M	1025580	2/10/2017	3/4/2017	25	H & N	42.39	42.16	83
83	ANBARASAN	79	M	1025600	2/13/2017	3/10/2017	28	H & N	37.16	36.42	88
84	NARAYANAN	45	M	1025609	2/23/2017	3/18/2017	26	H & N	39.41	38.87	87
85	HASARABEGUM	45	M	1025913	3/9/2017	4/7/2017	28	H & N	40.65	39.93	84
86	KANNAN	62	M	1026112	3/27/2017	4/23/2017	26	H & N	35.2	34.63	81
87	SULAIMAN	55	M	1026310	3/3/2017	4/31/2017	28	H & N	39.72	38.18	83
88	RAJAN	47	M	1026699	3/17/2017	4/14/2017	28	H & N	42.05	41.37	85
89	SUSAIRAJ	57	M	1027076	4/4/2017	5/4/2017	30	H & N	41.64	40.25	94
90	KISHORE	42	M	1027098	4/20/2017	5/11/2017	28	H & N	43.17	42.62	92
91	DHANU	50	M	1028951	4/10/2017	5/7/2017	28	H & N	39.72	39.25	88
92	AROKIYAMARY	55	F	1029034	4/19/2017	5/18/2017	30	H & N	40.11	39.9	89
93	IBRAHIM	48	M	1029103	4/1/2017	5/2/2017	32	H & N	42.6	41.96	88
94	PANDI	58	M	1029147	4/10/2017	5/7/2017	28	H & N	43.6	42.75	87
95	JEGANNATH	52	M	1029239	5/7/2017	6/9/2017	32	H & N	43.25	42.2	85
96	KARUPANAN	56	M	1029292	5/14/2017	6/12/2017	28	H & N	36.54	36.1	84
97	CHANDRASEKHAR	58	M	10293091	5/3/2017	6/212017	30	H & N	37.42	36.69	88
98	MADHINABEGUM	52	F	10293651	5/21/2017	6/13/2017	23	H & N	38.53	38.05	81
99	KAMALANATHAN	55	M	10293626	5/7/2017	6/5/2017	28	H & N	41.7	40.69	81
##	ARJUNAN	53	M	10293961	5/9/2017	6/9/2017	30	H & N	42.8	42.1	40

ABBREVATIONS USED

NPWT	:	negative pressure wound therapy.
HD	:	hydrodebridement.
DOA	:	date of admission
DOD	:	date of discharge
GRAN AREA:		area of granulation tissue.
SSG	:	Split Skin Graft uptake
B	:	Betadine
H and N	:	Hydrodebridement and NPWT

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ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.Christeena Indrani J.
Course	:	PG in MS., General Surgery
Period of Study	:	**2015-2018**
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	Prospective study on indigenous negative pressure wound therapy after innovative hydrodebriment in treatment of diabetic ulcer in GRH, Madurai.
Ethical Committee as on	:	21.04.2017

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary Chairman Dean / Convenor

Prof Dr V Nagaraajan
M.D., MNAMS, D.M., Dsc.,(Neuro), Dsc (Hon)
CHAIRMAN
IEC - Madurai Medical College
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